

showed the feet long, lanky, and abducted. There was no stiffness, and the arch was apparently low. The tubercle of the scaphoid was about three-fourths of an inch below the connecting line on each foot without weight-bearing. A diagnosis of moderately flaccid flat foot was made.

CASE VII.—W. H. Five years ago this boy began to limp. His ankles and wrists became swollen. Since then he has stumped about with his feet in the same condition, and has had much pain and swelling. Examination (Fig. 11) showed marked valgus, abduction, and pronation of the ankles, which were swollen and bluish. On palpation the tubercles of the scaphoid were found to be very low, about an inch below the connecting line. The peroneal tendons were tight. There was stiffness in all directions. The patient toed out when he walked. In this case I placed a metal mark on the tubercle and took a Röntgen picture, which proved that this prominent point on the contour was really the tubercle (Fig. 12). A diagnosis of extreme rigid flat foot was made.

Both these cases illustrate common types of flat foot and the relative significance of the depression of the scaphoid tubercle as suggesting the severity of the deformity.

It is, perhaps, worth while to call attention to a possible source of error. In very severe types of flat foot (as from infantile paralysis) the head of the astragalus twists so far inward as to suggest that its prominence is the tubercle of the scaphoid (Fig. 13). Experience and the study of the skeleton, however, will prevent the observer from being misled.

A FURTHER CONTRIBUTION TO MY "SIMPLE" METHOD FOR THE QUANTITATIVE DETERMINATION OF PEPSIN IN A GIVEN GASTRIC JUICE.

By H. ILLOWAY, M.D.,

OF NEW YORK,

FORMERLY PROFESSOR OF DISEASES OF CHILDREN IN THE CINCINNATI COLLEGE OF MEDICINE
AND SURGERY; FORMERLY VISITING PHYSICIAN TO THE JEWISH HOSPITAL, CINCINNATI.

It is a well-known fact and has been repeatedly demonstrated, by me among others, that the essential factor in the digestion of proteids is pepsin. It is, therefore, of the greatest importance in all cases of chronic gastric disease, especially that form attended with diminished or inhibited secretion of hydrochloric acid, to determine (a) whether pepsin is present, and (b) if so, whether it is present in normal quantity, in increased or diminished quantity, or (c) whether it is altogether wanting.

Various methods have been devised for this purpose, all more or less complicated and not adapted to the needs of the busy practi-

tioner. In 1905 I published¹ my method for the quantitative determination of pepsin found in a given gastric juice. In that article I claimed that my method is the simplest devised, and gives results that are perfectly reliable and in a manner readily and easily interpreted. Since then the application of the method in a much greater number of cases has, to me at least, substantiated all the claims made for it at the time, and has also brought to light some new facts which give it additional value. It is for these reasons that I bring this method once more to the attention of my colleagues.

Since the publication of my first paper two other methods,² making the same claims to clinical applicability, have been devised and published. The first is the method of Jacoby and Solms,³ based upon the fact that a cloudy solution of ricin⁴ in dilute hydrochloric acid becomes clear on the addition of a peptic ferment, or a solution thereof.

The technique of the method is as follows: 0.5 gram of the cheap ricin⁵ is dissolved in 50 c.c. of a 5 per cent. solution of sodium chloride, and filtered; 2 c.c. of this solution, which is somewhat turbid, is put into each of five test-tubes, and 0.5 c.c. of a decinormal solution of HCl added. The ricin solution becomes at once milky, opaque. The gastric filtrate obtained after an Ewald and Boas test breakfast is variously diluted according to the degree of acidity it is found to possess: The hyperacid, from 1 to 100 to 1 to 10,000; the normal (40 to 60), from 1 to 100 to 1 to 1000. If the gastric juice to be tested is, say, of the normal variety, a 1 per cent. dilution thereof is prepared by adding to 1 c.c. of the filtrate 99 c.c. of distilled water. To each of the test-tubes prepared as above, and numbered respectively 1, 2, 3, 4, 5, there is added of the boiled gastric juice (the ferments it contains are destroyed by boiling) to No. 1, 1 c.c.; to No. 2, 0.9 c.c.; to No. 3, 0.8 c.c.; to No. 4, 0.5 c.c.; to No. 5, 0 c.c. (none of the boiled filtrate is added). This done, there is further added of the 1 per cent. dilution (unboiled) to No. 1, 0 c.c. (nothing); to No. 2, 0.1 c.c.; to No. 3, 0.2 c.c.; to No. 4, 0.5 c.c.; to No. 5, 1.0 c.c. Each of the test-tubes contains now altogether 3.5 c.c., and we have dilutions of the filtrate, No. 2, 1 to 1000; No. 3, 1 to 500; No. 4, 1 to 200; No. 5, 1 to 100. No. 1, to which no ferment (that is, none of the 1 per cent. dilution) has been added, serves as a control tube. The tubes are now corked and placed in the thermostat and

¹ Einfache Methoden zur quantitativen Bestimmung der vom Magen ausgeschiedenen Enzyme, Archiv f. Verdauungskrankheiten, 1905, xi, 145.

² Other methods besides these have been devised since, but they are strictly laboratory methods.

³ Ztschr. f. klin. Med., vol. lxiv.

⁴ "Ricin," Kobert, Merck: Exceedingly poisonous agglutinin from the seeds of *Ricinus communis* L. and closely allied to the bacteriotoxins; yields about 25 to 30 per cent. ash. White Powder.—Sol.: 10 per cent. solution of sodium chloride.—Caution: Handle very carefully; small particles in abrasions, eye or nose, may prove fatal; intravenous injections of 3 grains (0.2) are fatal.—Merck's Index, 1907.

⁵ An impure preparation made by the Vereinigten Chemischen Farbwerke, Charlottenburg, Berlin.

allowed to remain therein for three hours; then they are taken out and examined to see which dilution has clarified the ricin solution. If a thermostat be not at hand, the procedure may be carried out at room temperature, but then requires a longer time. Having found on repeated examination that, of filtrates of normal acidity, 1 c.c. of a dilution of 1 to 100 (1 per cent.) just cleared the ricin solution in three hours, he called the pepsin content of this 1 c.c. 100 pepsin units, establishing it as his standard for comparison.

Witte⁶ has suggested certain modifications in this method. Having found in a series of cases of supposed normal acidity that the pepsin content of 1 c.c. of a dilution of 1 to 100 did not reach 100 pepsin units, he begins his dilutions of gastric juices of normal or slightly lowered acidity with 1 to 50. In cases of marked hypo-acidity or anacidity he may use the undiluted filtrate for the first tube and begin his dilutions with 1 to 1. As the HCl element varies greatly in quantity, he would have all gastric juices to be thus examined neutralized with a decinormal solution of potassium hydroxide and then brought to a uniform degree of acidity by the addition of decinormal solution of HCl.

Leaving out of consideration the question of the handling of a substance so fraught with danger, and, furthermore, leaving out of consideration the criticism of the method made by Gross and by Fuld and Levison, namely, that the ricin is full of impurities, and therefore all determinations based upon it are unreliable, a moment's thought will at once demonstrate that it is not a clinical method at all. The solutions and the many dilutions to be prepared, all of which, to give any value to the work, must be made with the greatest accuracy, a minute quantity more or less vitiating the result one way or the other, require more time and more care in their preparation than can be spared by the man in active practice who has a number of gastric juices to examine every day and other duties connected with his work to attend to.

Then, after all that painstaking work, what has the inquirer learned? He has learned that 1 c.c. of a certain dilution of a certain gastric juice is equal to the task of clarifying a certain solution of ricin. Does this reveal to him directly anything as to the digestive capacity of the stomach of the patient from whom the gastric juice was derived? Nothing at all. Only by a long stretch of the imagination and a long mathematical calculation based on figures that are essentially hypothetical (he even does not know the albumin content of the ricin he has used) could he form an idea of the possible digestive capacity of that particular stomach.

The second method is that of Fuld;⁷ Levison⁸ later published additional details. For his purpose he makes use of a 0.1 per cent. (1 to 1000) solution of edestin, a substance derived from hemp

⁶ Berl. klin. Woch., 1908, p. 643.

⁸ Amer. Med., February, 1908.

⁷ Münch. med. Woch., 1907, p. 1455.

and belonging to the category of the globulins. The edestin is dissolved in a solution of dilute hydrochloric acid having an acidity of 30 (corresponding, as he says, to the free HCl found normally after a test breakfast of Ewald and Boas). Six test-tubes (preferably of a diameter of 1 c.c.) are filled with various quantities of gastric filtrate obtained after an Ewald and Boas test breakfast and diluted according to its degree of acidity from 1 to 100 to 1 to 10 (for an-acidity). To each of these tubes 2 c.c. of the edestin solution is quickly added and the contents then allowed to digest for thirty minutes at the room temperature. At the expiration of the time named, strong ammonia is allowed to run down the sides of the tube until a layer is formed above the contents. The tubes are now examined by reflected light against a black background. If digestion is complete, nothing is seen, but if incomplete a distinct white ring of albumin is seen between the two layers—just as in Heller's test for urine.

By finding the test-tube in which a distinct ring ceases to be formed, the point where digestion is complete is determined, and by that the strength of the pepsin content of the given gastric juice. Thus, if the tube with 0.7 c.c. gastric filtrate has no ring, and the next tube with 0.4 c.c. thereof has a ring, it can be said that the gastric juice of that stomach has a pepsin strength of 0.7 in a 1 to 100 dilution. This can be recorded in another way: The dilution of the stomach contents is multiplied by the amount of the edestin solution used and the product divided by the amount of gastric filtrate placed in the tube; thus, 100×2.0 (read as 20), divided by 0.7, equals 285+. The pepsin value of that gastric juice is then said to be 285+.

Or the test can be carried out in this way: Instead of the ammonia, a 10 per cent. solution of sodium chloride is added to the tube. This will produce cloudiness proportionate to the degree of digestion that has taken place. The more ample the digestion, the lighter the cloudiness; in other words, the greater the quantity of the solution that will be required to render the contents of the tube cloudy. Thus, at the outset 0.5 c.c. of the salt solution may produce turbidity, while half an hour later it may require 2 c.c., or even more, to produce the same result. In this wise the test can be made with but a single tube.

It can be made in still another way: A 1 per cent. solution of edestin is prepared as above directed. Of this a sufficient quantity (5 or even 10 c.c.) is placed in a test-tube and digested with a not too strongly diluted gastric filtrate. At the expiration of half an hour solid sodium chloride is added, the tube well shaken, and the height of the precipitate measured. Levison says, in further explanation, that "the precipitated albumin very quickly assumes a definite volume, and as it exists under no considerable pressure, its volume is dependent upon the pepsin amount, and allows itself to be read off after standing a short time."

It will be at once apparent to the reader that this method also is not the simple one it is held out to be. All that has been said in criticism, in this one regard, of the method of Solms can be said of that of Fuld, namely, it is too complicated; the making of the principle solution, the dilution of the gastric filtrate, the filling of a number of tubes with minimal quantities which, to give reliable results, must be accurately measured (that it is not an easy matter to measure accurately 0.7 or 0.4 or 0.3 c.c. will be acknowledged by all laboratory workers, and is clearly shown by the numerous instructions given by the writer of the article as to the use of graduated tubes, pipettes with rubber bulb, etc.)—0.05 c.c. too much or too little, and the whole calculation goes wrong.

Then, after all the work has been done, what has the examiner learned? Nothing! His question, whether the gastric juice of a certain patient has enough pepsin content to carry on normally the process of digestion, remains unanswered.

A method to be clinical, that is, adapted to the needs of the man of large practice with not much time to spare, must be possessed of the following qualifications: (1) It must be simple in its technique, and every step thereof definite and accurately measured; and (2) it must give a result at once apparent, at once understood, without the necessity of long and complex calculations or of the translation by way of the imagination of hypothetical figures into those of a supposed actuality.

Of all the methods hitherto described, only the one devised by me measures up to this standard. It is as follows:

(a) Ten centigrams (exact weight) of egg albumen (white of egg*) is coagulated in the following manner: The egg is put into a pot of cold water, which is then covered with a lid and set on to cook. It is allowed to cook for ten minutes after the water has begun to boil—in all, twenty minutes from the time it was put on. The egg is then taken out and allowed to cool, either by just setting it out in a saucer or by putting it into cold water. Furthermore, to imitate the usual way in which food is or should be put into the stomach, the segment of albumen is divided into two parts. (Observation has shown—just as it has long been known clinically—that when it is thus subdivided the gastric juice can act upon it more quickly. The action being from all sides, it is naturally more effective when there are eight sides of attack for a given quantity than when there are but four.) (b) The two parts of coagulated egg albumen are put into 10 c.c. of the gastric filtrate from the stomach contents extracted one hour after an Ewald and Boas test breakfast; and (c) they are then placed in the thermostat, which is kept at 38° C. The time in which the 10 centigrams is digested entirely, partially, or not at all, will give us a correct idea as to the status of the pepsin secretion in the case under examination.

The results obtained with this method are well shown in the following tables:

* Hens' eggs only.

TABLE I—RAPID DIGESTION (less than the normal period.)

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I F.	N. K.	42	62	Hyperacidity	2.23	
II M.	A. A.	38	48	Hyperacidity, subacute gastritis, constipation	3.00	15 centigrams in 10 c.c. filtrate digested fully in 5½ hours.
III M.	F. H.	66	99	Hyperacidity, constipation	3.00	15 centigrams in 10 c.c. filtrate digested fully in 3½ hours.
IV M.	R. M.	56	77	Hyperacidity	3.10	
V M.	S. B.	62	77	Hyperacidity, constipation	3.10	
VI F.	M. B.	12	28	Deficient HCl; in the climacteric	3.15	Eats too heavy food. Her teeth are bad, and she cannot masticate well.
VII M.	A. E.	50	68	Hyperacidity, constipation	3.21	
VIII M.	J. K.	47	69	Eats too much and too heavy food	3.26	Adenoids and obstructed nose.
IX M.	A. K.	71	102	Hyperacidity and constipation	3.20	
X F.	B. C.	38	64	Hyperacidity, constipation	3.30	6 centigrams in 5.5 c.c. filtrate digested in 3½ hours.
XI F.	Y. G.	35	58	Constipation, inflamed hemorrhoids	3.30	
XII M.	J. F.	No reaction to phlor. gluc.v.	24	Constipation, colics, deficient HCl	3.30	With the addition of HCl c. p., gtt. ii. Without the acid at the end of 11 hours, the edges only slightly thinned.
XIII M.	J. L.	46	67	Constipation, hyperacidity	3.40	
XIV F.	E. A.	20	54	Constipation, dyspepsia	3.42	
XV M.	A. B. F.	40	57	Hyperacidity, constipation	3.40	His father suffered from hyperacidity, and finally died in an attack of hematemesis.
XVI M.	B. I.	29	58	Constipation	3.49	
XVII M.	B. G. C.	70	115	Hyperacidity	3.50	15 centigrams in 10 c.c. filtrate in 3 hours 50 minutes all digested except one small particle. That remained even after 4 additional hours.
XVIII M.	J. P.	30	54	Neurasthenia, constipation, hyperacidity	3.50	Three months later similar trial; 10 centigrams in 10 c.c. filtrate digested in 3 hours and 30 minutes.
XIX M.	N. W.	63	85	Hyperacidity, constipation	3.56	
XX M.	E. W.	41	59	Neurasthenia, hyperacidity	4.00	
XXI F.	S. M.	47	69	Hyperacidity, constipation	4.00	
XXII M.	E. M.	19	37	Atony of stomach	4.00	Lives in a warm climate. Eats food that is too heavy.
XXIII M.	L. R.	56	87	Constipation, hyperacidity	4.00	
XXIV F.	M. D.	22	54	Subacute gastritis	4.00	Had a gastro-enterostomy made on her before I saw her.
XXV M.	G. W. F.	39	71	Hyperacidity	4.09	
XXVI M.	I. B.	17	31	Constipation, slight deficiency in HCl	4.12	
XXVII F.	S. W.	27	57	Subacute gastritis	4.15	
XXVIII M.	L. B.	22	46	Constipation, secondary atony of stomach	4.15	
XXIX M.	A. W.	41	64	Hyperacidity, constipation, neurasthenia	4.15	

TABLE I.—Continued.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
XXX M.	I. C.	48	79	Hyperacidity, constipation	4.19	
XXXI M.	A. M.	50	61	Tubes. (beginning of) hyperacidity	4.20	
XXXII M.	M. A.	45	61	Hyperacidity, constipation	4.20	
XXXIII F.	S. H.	08	24	General debility, neurasthenia	4.20	
XXXIV M.	G. M.	38	64	Hyperacidity	4.25	
XXXV M.	Ph. M.	29	50	Constipation, uric acid gravel	4.25	
XXXVI F.	Mrs. B.	28	55	Hyperacidity	4.29	Determined from the symptoms, which are the same as in the most marked cases.
XXXVII F.	F. S.	28	51	Neurasthenia, constipation	4.30	
XXXVIII M.	D. H.	53	81	Hyperacidity, constipation	4.30	
XXXIX M.	M. S.	25	50	Subacute gastritis	4.30	
XL F.	D. B.	20	40	Subacute gastritis, diarrhoea	4.30	
XLI M.	N. P.	29	61	General debility, constipation	4.30	Taught school and studied law.
XLII M.	S. B.	22	45	Neurasthenia	4.54	Repeated a month later. Same results.
XLIII M.	H. C. B.	45	64	Hyperacidity, constipation	4.35	
XLIV M.	B. M.	19	34	Dyspepsia, constipation	4.40	
XLV M.	H. R.	36	59	Hyperacidity	4.40	
XLVI M.	H. G.	23	81	Hyperacidity, subacute gastritis	4.45	
XLVII M.	S. Gr.	24	58	Stricture of pylorus	4.45	

As already stated elsewhere,¹⁰ and for the reasons there given, I fixed upon 10 centigrams of albumen in 10 c.c. of filtrate, and five to five and one-half hours (at the farthest) to be taken as the normal standard for both quantity and time. Any deviation from this standard in either direction could then be readily recognized, and this deviation should then be, as it has been in all my cases, in harmony with the clinical history. That this method is correct for both quantity and time was borne out by the first series of cases reported,¹¹ and is fully confirmed by the tables here presented.

Table I shows a deviation downward. The 10 centigrams is digested more rapidly than usual, showing a greater digestive force in those particular gastric juices. This was proved in some of the cases (in those in which sufficient of the filtrate remained over for further examination) by a subsequent examination with 15 centigrams of the albumen to 10 c.c. of filtrate. In my first series of cases (coming under this same head), in all the instances in which a second examination with 15 centigrams was made the time required for the digestion of the greater quantity was the full normal time, five and one-half hours. In the present series we have three cases in which this examination

¹⁰ Archiv. f. Verdauungskrkh., loc. cit.

¹¹ Loc. cit.

was made. In Case II the time required for the digestion of the 15 centigrams was five and one-quarter hours, the normal time. It indicated that in this case the pepsin in the gastric juice was augmented by 50 per cent. beyond the normal.

TABLE II.—DIGESTION IN NORMAL PERIOD.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I F.	L. H.	32	53	Constipation, hyperacidity	5.00	
II M.	M. C.	45	67	Hyperacidity, subacute gastritis, constipation	5.00	Suffers much with cramps.
III M.	M. J. C.	17	44	Constipation, dyspepsia	5.00	
IV F.	R. H.	24	48	Constipation; impaired respiratory activity	5.00	Nose very much obstructed.
V M.	S. B.	25	40	Stricture of œsophagus	5.00	
VI F.	L. C.	21	51	Constipation, colics	5.00	
VII M.	L. G.	26	60	Constipation	5.00	The constipation due to a wrong diet.
VIII F.	L. T.	19	40	Constipation	5.02	
IX M.	I. S.	84	98	Hyperacidity	5.03	
X M.	C. B.	35	65	Hyperacidity	5.05	
XI M.	J. H.	59	84	Hyperacidity, constipation	5.05	
XII M.	M. W.	26	64	Colics	5.09	
XIII F.	A. M. H.	28	56	Dyspepsia	5.10	
XIV M.	W. S.	25	40	Subacute gastritis, gastrosuccorrhœa	5.10	Subsequently obstruction of bile duct and jaundice.
XV M.	M. A.	46	62	Bright's disease	5.12	
XVI M.	C. N.	25	48	Hyperacidity, dyspepsia, constipation	5.12	
XVII M.	S. A. V.	37	53	Hyperacidity, subacute gastritis	5.13	
XVIII F.	N. D.	34	46	Constipation, tendency to hyperacidity	5.18	
XIX F.	Mrs. Z.	30	69	Neurasthenia	5.20	
XX F.	F. G.	30	45	Bad mastication, bad teeth, constipation	5.20	
XXI M.	A. B.	33	59	Tendency to hyperacidity, irritability of stomach, nervous	5.24	
XXII M.	S. R.	41	61	Hyperacidity	5.24	Nervous temperament.
XXIII M.	M. B.	34	59	Hyperacidity, constipation	5.30	
XXIV F.	F. R.	13	69	Constipation, dyspepsia	5.30	
XXV M.	F. B.	24	45	Rheumatism of stomach, subacute gastritis	5.30	Tendency to hyperacidity.
XXVI M.	M. G.	44	76	Hyperacidity	5.30	Much coffee.
XXVII F.	A. R.	19	39	Constipation, deficient HCl, nervousness	5.30	Leads an irregular life.
XXVIII M.	C. J. L.	655	25.5	Deficient HCl, irritability of intestinal tract	5.30	
XXIX M.	A. E.	30	52	Subacute gastritis	5.30	
XXX M.	S. J. P.	59	81	Hyperacidity, slight subacute gastritis	5.30	

In Case III the 15 centigrams was digested in three and one-half hours, showing that in this case the quantity of pepsin secreted was greater even than in Case II, possibly double the normal. I regretted

TABLE III.—DELAYED DIGESTION (more time than the normal period required).

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion.	Remarks.
I M.	M. P.	31	51	Constipation, tendency to hyperacidity	5.50	At the time of examination a question as to whether he had had a hepatitis shortly before or not.
II F.	M. M.	28	44	Constipation, secondary atony of stomach	6.00	
III F.	L. P.	22	51	Chronic diarrhoea	6.00	Has lost very much flesh. The symptoms pointed thereto (hyperacidity) and the dietary directions for this same brought rapid relief.
IV M.	M. L. R.	26	71	Hyperacidity, constipation, angina pectoris	6.10	
V F.	N. B.	03	37	Chronic gastric catarrh	6.10	
VI M.	M. S.	25	50	Hyperacidity, pseud o-angina pectoris	6.10	
VII F.	L. K.	05	26	Defective gastric juice, costive	6.20	
VIII F.	F. D.	10	32	Defective gastric juice, constipation	6.20	
IX F.	P. W.	14	27	Slight catarrh of the stomach	6.20	
X M.	F. D.	13	45	Defective gastric juice, constipation	6.30	
XI M.	J. C. L.	10	29	Defective gastric juice, constipation	7.00	
XII M.	L. M.	45	65	Hyperacidity, constipation	7.03	
XIII F.	L. K.	05	28	General impairment of the digestive organs reflexly from ailments of the genital tract.	7.15	
XIV F.	B. K.	No reaction to Congo, none to ph. gluc. v.		Chronic gastric catarrh, tendency to epileptic seizures.	10.20	

very much that for lack of filtrate I could not carry the examination farther, to 17.5 centigrams, and if this was digested in less than the normal time, to 20 centigrams.

In Case XVII the greater part of the 15 centigrams was digested in three hours and fifty minutes; a small particle remained over and was still there and as large after four hours more in the thermostat. This demonstrated that the digestive capacity of these 10 c.c. of filtrate was beyond 10 centigrams, but not quite up to 15. Here also the lack of filtrate prevented a reëxamination with a less quantity, namely, 13 centigrams (the particle remaining over in the second examination I judged to be about 2 centigrams).

TABLE IV.—INSUFFICIENCY OF PEPSIN (in all these cases the heating was continued for 4 or 5 hours more after the time named, but no further digestion was effected).

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion and remarks.
I M.	F. R.	43	69	Hyperacidity, constipation	In 3 hours 39 minutes all digested except two or three minute particles.
II F.	B. B.	23	69	Stricture of pylorus (benign)	In 4 hours all digested except a number of minute particles.
III M.	J. H. C.	47	72	Hyperacidity, constipation	In 4 hours 10 minutes all digested except one fine scale. At the end of 7 hours scale still present.
IV M.	S. L. J.	48	71	Hyperacidity, cosiveness	In 4 hours 30 minutes all digested except three minute particles.
V F.	L. S.	16	37	Deficient HCl, constipation	In 4 hours 40 minutes all digested except a few minute particles.
VI F.	Mrs. H.	14	51	Constipation, flatulence	In 5 hours all digested except two minute particles.
VII M.	B. S.	73	93	Hyperacidity	In 5 hours all digested except one particle.
VIII M.	M. C.	41	60	Hyperacidity, constipation	In 5 hours all digested except two minute particles.
IX F.	L. S.	08	27	Defective gastric juice, constipation	In 5 hours all digested except one minute particle.
X F.	L. J.	15	38	Gastralgia, constipation, defective gastric juice	In 5 hours all digested except a few minute particles. Five centigrams in 10 c.c., all digested in 3 hours 30 minutes.
XI M.	L. B.	32	58	Hyperacidity, constipation	In 5 hours 7 minutes all digested except two or three particles.
XII F.	V. B.	21	51	Cystic tumor of right ovary	In 5 hours 7 minutes all digested except four or five minute particles.
XIII M.	J. G.	48	64	Hyperacidity	In 5 hours 25 minutes all digested except one faint scale.
XIV M.	H. G.	77	96	Hyperacidity, constipation	In 5 hours 40 minutes all digested except a few minute particles.
XV F.	A. C.	12	34	Defective gastric juice, constipation	In 6 hours all digested except three or four particles.
XVI F.	A. R.	12	42	Defective gastric juice, constipation	In 6 hours all digested except three or four minute particles.
XVII F.	R. L.	27	50	Status epilepticus	In 6 hours 29 minutes all digested except two minute particles.
XVIII F.	H. H.	06	21	Defective gastric juice, constipation	In 6 hours 30 minutes all digested except one small piece, thinned at edges only.
XIX M.	W. K.	55	70	Hyperacidity	In 6 hours 35 minutes all digested except two small particles.
XX M.	J. M.	53	72	Hyperacidity, nervous	In 6 hours 41 minutes all digested except two particles.
XXI M.	D. U.	41	65	Neurasthenia, hyperacidity	After 7 hours a scale of one segment (about one-third of it) remained undigested.
XXII F.	F. J.	10	58	Defective gastric juice, atony of stomach, hyperesthesia of intestinal mucous membrane	After 7 hours 13 minutes a large part of the one segment (though borders were very much thinned down) and a large scale of the other segment remaining over.
XXIII F.	Mrs. H.	13	28	Defective gastric juice, constipation, irregular eating, rich food	After 7 hours 15 minutes a very small particle and several minute ones over.
XXIV M.	L. D.	11	32	Defective gastric juice, slight catarrh of stomach	After 7 hours 30 minutes a number of minute particles still present.
XXV F.	J. C.	27	51	Chronic ovaritis, chronic appendicitis	After 7 hours 45 minutes a number of minute particles still present.
XXVI F.	J. S.	32	49	Atony of stomach	After 7 hours 48 minutes two small scales still present.
XXVII M.	M. R.	29	43	Constipation	After 8 hours several minute particles still present.
XXVIII M.	J. F.	34	63	Hyperacidity, atony of stomach second degree, atony of bowels	After 8 hours one small particle and several minute ones still over. (Test repeated; same result.)

TABLE IV.—Continued.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Hours of digestion and remarks.
XXIX F.	Miss L.	14	43	Defective gastric juice, atony of stomach	After 8 hours all digested except one thin scale.
XXX M.	S. J. P.	26	41	Gastralgia	After 8 hours several small particles still present.
XXXI F.	U. J. K.	02	..	Chronic gastric catarrh	After 8 hours 30 minutes 2.5 centigrams over.
XXXII M.	W. A. J.	58	77	Hyperacidity	After 8 hours 40 minutes two or three particles still present.
XXXIII M.	J. B.	43	78	Hyperacidity (bad teeth, bad mastication)	After 8 hours 43 minutes four small particles still there.
XXXIV M.	S. P.	21	47	Gastralgia	After 9 hours a number of minute particles still present.
XXXV M.	S. E.	17	49	Sarcoma of intestines	After 10 hours a scale of each segment left.
XXXVI F.	R. B.	16	26	Gastralgia, constipation	After 10 hours a minute particle still present.
XXXVII M.	J. W.	21	37	Beginning gastric catarrh	After 10 hours 30 minutes two or three minute particles still present.
XXXVIII B. Th.		40	52	Hyperacidity, constipation	After 11 hours a minute scale still present.
XXXIX A. K.		09	39	Defective gastric juice, constipation	After 11 hours two or three minute particles still present.
XL F.	J. R. W.	10	41	Defective gastric juice	After 12 hours three minute particles still over.
XLI F.	B. K.	10	28	Chronic gastric catarrh	After 13 hours four minute particles still present.

TABLE V.—VERY LITTLE DIGESTION OR NONE AT ALL.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Time.
I F.	C. T.	No reaction to Congo	..	Chronic gastritis	10 centigrams in 10 c.c. filtrate + gtt. ii HCl c.p. After 9 hours unaffected.
II M.	E. N.	Reaction: blue litmus + Congo — resorcin —	..	Chronic gastro-intestinal catarrh	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl gtt. ii c.p. After 11 hours digested down to one large scale and several small particles (in all about one-third left.
III F.	S. W.	Reaction: blue litmus — Congo —	..	Cirrhosis ventriculi	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl c.p. gtt. ii, no effect.
IV F.	S. R.	Reaction: Congo + filtered Congo — ph. gluc. v. —	18	Neurasthenia, constipation	10 centigrams + 10 c.c., no effect. 10 centigrams + 10 c.c. + HCl c.p. gtt. ii, digested in 6 hours.
V M.	N. S.	Reaction: blue litmus + Congo —	..	Nervous prostration	Without HCl, no effect; with HCl, digested all but one particle in 6 hours.
VI M.	A. St.	Reaction: blue litmus + Congo —	..	Cirrhosis ventriculi	10 centigrams with HCl gtt. ii at end of 5 hours untouched.
VII M.	H. W.	Reaction: blue litmus + Congo —	..	Cirrhosis ventriculi	10 centigrams with HCl at end of 10½ hours untouched.
VIII M.	N. R.	Reaction: blue litmus + Congo —	..	Chronic gastric catarrh	10 centigrams without HCl after 10½ hours no digestion at all. With HCl at end of 10½ hours one large scale and a few particles over, altogether about 3 centigrams left over.
IX F.	S. F.	Reaction: blue litmus + Congo —	..	Chronic gastric catarrh	Without HCl, unaffected. With HCl at end of 8 hours, one segment slightly thinned at edges.
X M.	S. D.	0.1	2.2	Chronic gastric catarrh	Without HCl after 9 hours and 7 minutes, the four borders of each segment thinned down, translucent almost. With HCl, all digested in 9 hours and 7 minutes, except 2 small fine scales.
XI F.	J. V.	Filtered. No reaction to ph. gluc. v.	3.0	Constipation, neurasthenia	Without HCl, not affected. With HCl at end of 5 hours and 7 minutes all digested except two minute particles.

The points of special interest in this table are the indications (a) that in many cases of hyperacidity, perhaps in the greater number, there is also an increased secretion of pepsin,¹² and (b) that it is not by the excess of acid that the digestion is hastened. Thus, in Case XIV, with a degree of acidity, both free and combined, considerably lower than many of the others that follow, the digestive act was complete in much less time than was required, say, in Case XVII, with more than three times the quantity of free acid and more than double the quantity in the total acidity. The same can be seen in a number of other cases in this same table, and it is very clearly shown when comparisons are made with some of the cases of hyperacidity (Cases IX, XI, and XXVI) of Table II, and most strikingly so when the comparisons are made with some of the cases of hyperacidity of Table III (Cases IV and XII) and of Table IV (Cases IV, VII, and VIII).

There is yet something more that this table seems to disclose. It is the possibility that just as there may be an increased secretion of HCl without a corresponding increase in the secretion of the pepsin, so there may be an increased secretion of pepsin without any corresponding increase in the secretion of HCl. Cases XIV, XXII, XXVI, and XXXIII seem to point thereto.

In Table II the cases all conform to the standard in quantity and time. Here also we have a very interesting observation, the corollary of that noted above, namely, that there may be an increased secretion of HCl without a corresponding increase in the pepsin.¹³ The patients with hyperacidity of Table I had, with all their complaints, a fair to good appetite, and could, when properly directed as to diet, eat again four hours after a hearty meal; those of Table II did not have that early hunger—in fact, some of them complained of poor appetites. In some of the cases of this table the greater part of the 10 centigrams was digested in four hours or a little over; still, as long as a speck remained, the act was not counted complete.

Tables III, IV, and V show us the deviations upward, there being a deficiency in the quantity of pepsin secreted. In Table III we note a deficiency; the time required for a completion of the process of digestion is more or less greater than the normal time. Nevertheless, the deficiency can be but slight, as there is still enough pepsin secreted to digest completely the test quantity. In Table IV the deficiency is greater, and part of the test quantity remains undigested. How much this is can be readily estimated either by simply judging it by the eye, which is rather unscientific, or by the more accurate method of removing it from the beaker to a filter and weighing it.

Still another fact of great interest is presented to us by these tables, namely, that we may have hyperacidity associated with a deficient

¹² This was first definitely demonstrated by my tables.

¹³ *Ibid.*

secretion of pepsin, from the minimal deficiency of Table III, causing only a protraction of the act of digestion, *i. e.*, requiring a longer time for its completion, to the larger one of Table IV, entailing a marked diminution of the digestive capacity of that gastric juice.

In Table V we observe a total or almost total absence of pepsin and pepsinogen. Here we have a further demonstration of the fact that the HCl constituent alone does not effect digestion. In Cases I, III, VI, and VII pepsin and pepsinogen were altogether wanting, and the addition of sufficient HCl (*c. p.*) to give free acid 22 did not in any way change the result.

To summarize, it may be claimed for this method:

1. That it is simple and eminently practical.
2. That from the simplicity and exactness of its preliminaries it is well adapted for comparative study.
3. That it is directly demonstrative, in that the examiner has a direct ocular demonstration, as it were, of the digestive capacity of a given gastric juice, and can thus at once, without any long mathematical calculations, which not infrequently have only a fictitious basis, determine whether in the case under examination there is:

Hyperpepsinia: Requiring only three to four hours for digestion. Not necessarily in a pathological sense, only to indicate a secretion of pepsin greater than usual, which may, however, be normal for that case.

Normal pepsinia: Requiring only five to five and one-half hours (the normal period) for digestion.

Hypopepsinia: Requiring more than the normal time for digestion (deficiency in the pepsin secretion).

Apepsinia: No digestion at all (total absence of pepsin and pepsinogen).

TABLE VI.—DIGESTION IN LESS THAN THE NORMAL PERIOD. Less than 10 c.c.

Number of case and sex.	Name.	Free HCl.	Total acidity.	Diagnosis.	Quantity.	Time.
I M.	E. G.	45	85	Hyperacidity	4 centigrams to 4 c.c. filtrate	3.55
II F.	P. K.	11	22	Deficient HCl, constipation, nervousness	5 centigrams to 5 c.c. filtrate	3.55
III M.	J. L.	42	74	Hyperacidity, intestinal colic	5 centigrams to 5 c.c. filtrate	3.30
IV F.	B. C.	38	64	Hyperacidity, constipation	6 centigrams to 5.5 c.c. filtrate	3.30
V M.	J. St.	65	92	Hyperacidity, constipation	7 centigrams to 7 c.c. filtrate	3.35

But a mathematically still more closely defined result can be obtained with this method. In the course of this work it occurred to me, and the reasons therefor are self-evident, that possibly each of the 10 c.c. possessed the power of digesting individually 1 centi-

gram of the white of egg above described. A number of trials were made, and the results proved this to be so.

Additional confirmation was afforded by the results obtained in some other cases:

In Case X of Table IV the 10 c.c. were not able to digest the 10 centigrams entirely; a few minute particles remained over, and allowing the beakers to remain in the thermostat for four or five hours more than the normal time did not change the result; but 5 centigrams was digested by 10 c.c. in three and one-half hours, two hours less than the normal time. It was clearly apparent, therefore, that though the 10 c.c. was not quite equal to 10 centigrams (in other words 1 c.c. to 1 centigram), it was more than equal to 5 centigrams (or 1 c.c. to more than 0.5 centigram). If more filtrate had been left the examination could have been continued until the exact limit for quantity had been reached; judging by what was left over on the first trial, I should say that this would have been about 9 to 9.5 centigrams.

F. A. G., reaction to blue litmus +	6 centigrams in 6 c.c. after 9 hours 15 minutes, edges only slightly thinned.
reaction to Congo +	6 centigrams in 6 c.c. + HCl (c. p.) gtt. ii. Complete digestion in five hours.
Filtered, reaction to Congo —	

Here, when we have the pepsin activated by the addition of HCl, we get a striking verification of our proposition. It can, therefore, be said that the normal quantity of pepsin in the ordinary normal stomach is equivalent to the digestion by each cubic centimeter of the gastric juice of 1 centigram of white of egg, prepared as above described, in from five to five and one-half hours.

ADENOFIBROMA OF THE MALE BREAST.

By R. T. WOODYATT, B.S. M.D.,
OF CHICAGO.

(From the Pathologic-Anatomical Institute of the University of Strassburg,
Prof. Chiari, Director.)

In March, 1907, there was sent to the laboratory a tumor mass which had been extirpated from the breast of a boy. This was put into my hands for study by Prof. Chiari. It had a fibro-epithelial structure corresponding to that of ordinary adenofibroma, such as may occur in the female breast. In attempting to ascertain the frequency of such growths in the male breast some difficulty was encountered, because of the uncertainty which still prevails in the literature